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Serum Lead:
Relation between Maternal and Cord Levels

AMINA M. ABDEL WAHAB, M.D.; MEDHAT ABOUT ZIED, Ph.D;
MOHAMMED I. EL KALIOBY, M.D.;
NASHWA M. EL SHINAWY, M.D. and NADIA M. GAZAR, M.D.

The Departments of Pediatrics and Obstetrics & Gynecology, Faculties of Medicine, Suez Canal and Al-Azhar Universities and the Department of Pharmacology, NCI, Cairo University.

Abstract

This study comprised 51 full term newborn babies as well as their low socioeconomic mothers. The cord serum lead levels of the born babies together with the maternal serum lead levels were determined. The cord serum lead level ranged from zero to 257 µg/dl with a mean ± standard error 34 ± µg/dl, while the maternal serum lead level ranged from zero to 429 µg/dl with a mean ± standard error 84 ± 13 µg/dl. A strong relation between maternal serum lead level and cord serum lead level was deduced. With zero level in maternal serum, the corresponding value of their babies was always zero while with level 1 - 99 µg/dl in mothers' serum 36% of babies showed detectable lead in their serum. With the increase of lead level in mothers' serum, a large percentage of babies showed high level in their serum. This study highlights the great danger the newborns were subjected to; the high level of maternal serum lead that reflected exposure to lead environmental pollution represented a real danger to the health of their babies.

Introduction

LEAD is virtually ubiquitous in the environment [1, 2] as a result of its natural occurrence and its industrial use. The use of lesser amounts of lead (e.g., tetraethyl lead) in gazoline during the past decade has resulted in a decrease in blood lead concentrations in man [3]. Overall, human exposure to lead is primarily from food. Most of the overt toxicity from lead results

from environmental and industrial exposure. Although lead is not required for any physiologic process, lead readily binds to cellular constituents. At low lead concentration such binding may produce measurable physiologic effects. However, at increased concentration the normal biochemical processes are influenced which may in turn produce adverse effects. Low lead levels (i.e., 16 μg/dl) disrupt enzyme systems particularly heme synthesis [2]. Children whose blood lead levels exceed 20 μg/dl begin to exhibit deficits in neurologic performance [4, 5]. Higher blood lead levels (i.e. 80-100 μg/dl) can result in encephalopathies and death [6]. The fetus is also at high risk from lead exposure; in fact lead had been used as an abortifacient. Bellinger et al. [7] demonstrated a measurable alteration in behaviour at 1 and 2 years of age in a cohort of infants whose average cord blood lead level was 14.6 μg/dl. They hypothesized an in utero effect of lead on the embryo's neurological development. In 1988, a report on lead poisoning in children presented by the Agency for Toxic Substances and Diseases Registry (ATSDR) concluded that prenatal exposures that result in blood lead levels of 10-15 μg/dl are associated with deficits in neurobehavioral development, aminolevulinic acid dehydratase inhibition and reduced gestational age and weight at birth [8].

Current evidence indicates that lead may produce adverse health effects at levels below the Centers for Disease Control (CDC) cut off (i.e. 25 μg/dl) [9] for a safe body lead burden. This coupled with the fact that these effects may begin during exposure in utero provide a cause for concern.

This study was conducted to determine cord serum as well as maternal lead levels in some low socioeconomic Egyptians and the relation between both levels to highlight the magnitude of the problem.

Subjects and Methods

This study was conducted from July to September 1993 on 51 randomly selected newborns and their mothers attending the Obstetric Department of el Zahraa University Hospital for delivery. Full history was taken from the mothers with special stress on the residence, occupation, the family members, the housing environment and cooking utensils. Also, full obstetric history was taken with special stress on the parity and the outcome of previous pregnancies. Full clinical examination was performed for the mothers including weight, height, blood pressure and full obstetric examination.

After delivery, babies were subjected to thorough clinical examination with stress on the weight, height, skull circumference, the Apgar Score, assessment of gestational age and search for congenital anomalies.

Approximately 10 ml of cord blood were collected in blue topped lead free

sterile tubes. Samples were collected after umbilical cord had been severed under complete aseptic precautions. This is in addition to blood samples from the mothers taken after clinical examination. The samples were collected under complete aseptic precautions.

The serum lead content was quantitatively determined directly using Atomic Absorption Spectrophotometer SP 1900. PYE UNICAN Ltd. Cambridge, England.

Results

This study included 51 newborn infants; 27 males and 24 females. Twelve babies were delivered by cesarean section for malpresentation while the rest had normal vaginal delivery. All babies were full term and all of them were assessed for vital signs which came to be within normal limits. Physical examination of all babies revealed no congenital anomalies apart from 2 babies who had small head circumference (head circumferences 30, 31 cm respectively) mostly due to overriding of skull bones. Their weight ranged from 2.400 Kgm to 4.100 Kgm with a mean of 3.300 ± 455 gm.

The age of the mothers ranged from 18 to 42 years with a mean of 25.3 ± 6.9 years.

All the mothers were of low socioeconomic class, 15 were multigravida. None of them had complications during pregnancy. They had no special habits apart from 2 who were moderate smokers (less than 10 cigarettes per day). They were of average built and no abnormality was detected on physical examination.

Cord serum lead level ranged from zero to 257 µg/dl with a mean of 34 ± 8 µg/dl (mean ± SE), while the maternal serum lead level ranged from zero to 429 µg/dl with a mean of 84±13 µg/dl. Fig. (1) shows the relation between maternal and cord serum lead level.

Fifteen mothers were primigravida, with a mean serum lead level of 66 ± 27 µg/dl, while 36 were multigravida with a mean of 87 ± 15 µg/dl. The difference between lead

Table (1): The Relation between Maternal and Cord Serum \ Lead Level.

Maternal lead level (µg / dl)	No. of mothers	Cord serum lead level (µg / dl)	% of babies showing lead level
0	13	0	0
1 - 99	25	15 ± 5	36
100 - 199	7	65 ± 21	71
200 - 299	4	120 ± 51	75
300 -	2	114 , 86	100

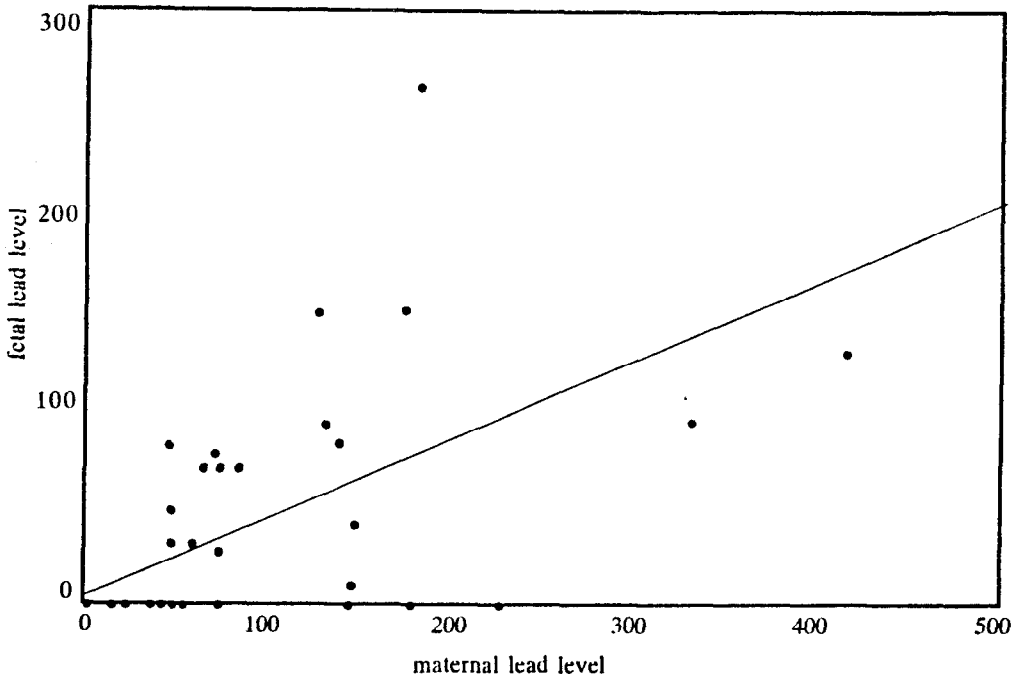

Fig. (1): The relation between maternal and cord serum lead level.

levels in the 2 groups was not statistically significant. The levels in their babies were 25 ± 10 µg/dl & 33 ± 10 µg/dl respectively. The difference between them did not rank to the level of statistical significance.

Discussion

Lead is present everywhere in the environment. Most of human exposure to lead results from food [3]. Acidic food and beverages including tomato juice, fruit juice, cola drinks, cider and pickles can dissolve lead in improperly glazed containers [8]. Lead poisoning in children is fairly common and results from ingestion of paint chips from old buildings. Sporadic cases of lead poisoning have been traced to miscellaneous sources such as lead toys, lead dust in shooting galleries, drinking water that is conveyed through lead pipes; hot water may be of particular concern. Lead contaminated water has been linked to lead poisoning in children given recenstituted infant formula [10]. Artists paint pigments, ashes and fumes of painted wood, jewelers, wastes and home battery manufactures are common sources of pollution. Occupational exposure to lead has decreased markedly over the past 50 years because of the appropriate regulations and programs of medical

surveillance. Lead may also contaminate food. Soil lead is taken up by roots of vegetables and atmospheric lead may fall into leafly vegetables [11]. Eye cosmetics (kohl used by Moslems) contain lead.

Lead is absorbed by ingestion or inhalation. The relationship between exposure and blood lead level is a dynamic process in which blood lead represents a product of recent exposure, excretion and equilibration with other tissues. Children deficient in iron, protein, calcium and/or zinc absorb lead more readily [12].

In this study, the mean maternal serum lead level at the time of delivery was 84 ± 13 µg% while the mean cord serum lead level was 34 ± 8 µg%.

One of the interesting findings observed in this study was that 13 newborn had zero lead level in their cord serum determination, their mothers had zero lead level also in their serum determination. The level of the serum lead in the newborn depended upon serum lead level in the mother, i.e., the higher the level of serum lead in the mothers the higher the lead level of the babies. However, the higher the level of the mothers serum lead the higher was the percentage of babies that showed lead levels in their serum.

Thirty six per cent of the babies whose mothers had serum lead level of less than 100 µg% had manifested level of lead in their serum, while 71% of the babies whose mothers had serum lead level between 100-199µg% had manifested lead in the serum. The percentage raised to 75% µg% and more than 300 µg%. These results suggested a strong direct relation between maternal and fetal serum lead level. ($r = 0.621, p < 0.001$).

The same observation was reported in a prospective study performed by Dietrich et al., [13] for children born to 305 women in the inner city of Cincinnati; prenatal (maternal) blood lead level was 8 µg/dl while neonatal levels averaged 4.5 µg/dl. Nearly the same correlation could be emphasized by Ernhart et al [14] in Cleveland Study determining the whole blood lead level instead of serum level. The mean blood lead level for 185 mothers was 6.5 µg/dl & for 162 umbilical cord samples the average was 5.8 µg/dl.

However, the results of these studies were much lower than that found in the present study. This can be attributed to the effect of the low socioeconomic level in our studied population compared to the soioeconomic standard in the well developed countries in the other reports. Our studied population are living in crowded areas where ventilation is bad and air pollution is expected to be at its maximum with the use of leaded fuel. Also house paintings are expected to be old ones and pipes water supply are other accused sources for contamination. This is in addition to the unawareness of the hazards of lead, sources of pollution and the means of its prevention.

The low socioeconomic status is usually associated with increased lead exposure especially from sources other than air e.g. occupation, soil and residential paint. Satin et al. [15] reported in their study no association between cord blood lead level with respect to ambient air lead gradient. However, it has been suggested that lead stored in maternal bones due to repeated previous exposure can be released during pregnancy and transfered to the fetus [16]. This could account for lack of association between cord and air lead level. Air born and other routes of exposure that influence water and food supplies as well as the maternal release of stored lead are probably more important for adult women and fetuses. Satin et al. [15] reported mean cord lead level that was approximately one fourth that of CDC cut off level for a "safe" body burden and highest value was only slightly greater than one half the CDC cut off. At the cut off level of 10 μg/dl neurological and behavioral decrements were observed.

Bellinger et al. [7] reported that cord blood lead level is strongly correlated with concurrent lead level in mothers' venous blood. The mothers' lead level appears either to remain stable or to decline slightly over the course of pregnancy. They reported lead level in umbilical cord blood in urban areas in the range of 7 - 9 μg/dl and concluded that the current standard of the CDC for acceptable lead levels in young children (< 25 μg/dl) should not be applied to fetuses. Obviously this shows up the magnitude of the danger our newborns, especially in the low socio economic areas, are subjected to.

In 1987, the American Academy of Pediatrics stated that lead levels greater than 25 μg/dl were unacceptable for children. The Academy now recognizes that impairment of cognitive function begins to occur at levels greater than 10 μg/dl even though clinical symptoms are not seen. The mean blood lead level of US Children has declined sine 1976 due to the phase our of lead in gazoline and the reduction of lead in food [3].

When we compare this US acceptable lead level and serum lead level in this study we could suggest the major danger of impairment of the cognitive functions in such babies.

Several studies have confirmed the association between prenatal lead levels and scores on Bayley infant development scales [17,18]. Many studies confirmed the possible importance of the 24 months lead level on cognition in school age children [19, 20]. Exposure during the first 2 years incurs a risk of sustained developmental delays and impaired cognition. Recent evidence suggests that the effects of early exposure can persist. Moreover, many investigators reported a link between prenatal lead exposure and impaired performance and the Baley Mental Development index (MDI) at 6 months

of age. Postnatal lead levels showed no association with MDI deficity nor did Byaley psychomotor development [21].

Needleman et al. [22] followed up a group of children who were identified by definite high level of lead in their first and second grade into adulthood. This study showed that those with high lead levels were seven times more likely not to graduate from high school and six times more likely to have reading scores at least 2 grades below expected after adjustment for a number of factors including socio economic status and parental IQ. The children also had higher absentism in the final year of school, lower class rank, poorer vocabulary and grammatical reasoning scores, longer reaction times and poorer hand eye coordination. Lansdown et al. [23] found that lead was associated with high scores on the overactivity factor of the Rutter B2 Scale and on the conduct problem, in attentive passive, and hyperactive scales of the Connars' Questionaire and Tailor-Sandhberg Hyperativity Score.

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